Case Reports

Sonographically Documented Disappearance of Nonimmune Hydrops Fetalis Associated With Maternal Hypertension

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NONIMMUNE HYDROPS FETALIS is a condition of the fetus characterized by varying degrees of anasarca, ascites and pleural and pericardial effusions in a pregnancy without rhesus isoimmunization. Diagnostic ultrasonography has permitted in utero diagnosis of this condition, which is frequently incidental to the original indications for the procedure. We report a case of nonimmune hydrops fetalis associated with maternal pregnancy-induced hypertension that spontaneously resolved over a period of several weeks, coincidental to the resolution of the pregnancy-induced hypertension.

Report of a Case

The patient, a 22-year-old gravida 2, para 1 woman with a previous normal term pregnancy, was having an uncomplicated pregnancy until 28 weeks' gestation when her blood pressure rapidly increased to 134/90 mm of mercury from a level of 110/60 mm of mercury at 12 weeks' gestation. Analysis of her urine showed 1+ protein and she had a 3+ pedal edema. Over the four weeks since her previous office visit, the patient's weight had increased from 56 to 65 kg (123 to 143 lb). Mild polyhydramnios was suspected by a rapid increase in fundal height. Ultrasound examination confirmed the polyhydramnios and showed placental thickening (8 cm), fetal ascites and pleural effusions (Figure 1). The maternal blood type was O Rh positive, with a negative screen for irregular antibodies. VDRL was nonreactive. The rubella titer was 1:80, the toxoplasmosis titer was less than 1:8 and testing for hepatitis B surface antigen was negative. Her hematocrit was 41%. A 24-hour urine collection showed a creatinine clearance of 78 ml per minute and a total protein of 289 mg per 24 hours. The serum albumin level was 2.8 mg per dl.

Over the next two weeks, evidence of maternal pregnancy-induced hypertension cleared with bed rest. In the same time period, ultrasound examinations documented the spontaneous clearing of the nonimmune hydrops fetalis with reabsorption of the effusions, ascites and polyhydramnios (Figure 2). Non-stress testing initiated at the time of the diagnosis of the hydrops fetalis and continued until delivery at 37 weeks consistently showed a reactive fetus without abnormal baseline heart rate or periodic changes. After a spontaneous labor, the patient was delivered of a vigorous 3,300-gram male infant with a cord blood hematocrit of 56% and evidence of a mild hepatitis. The cord blood serum alanine aminotransferase (formerly SGOT) was 74 units per liter (normal to 20) and the lactic dehydrogenase level was 585 units per liter (normal 100 to 225). Total bilirubin level was 9.2 mg per dl (normal 1 to 4) and the IgM was 43 mg per dl (normal 6 to 25). Viral culture of a urine specimen was negative for cytomegalovirus. Cultures of specimens from the throat were negative for adenovirus, enterovirus, echovirus and respiratory syncytial and herpes simplex viruses. The placenta weighed 1,000 grams but was unremarkable on histopathologic examination. The infant remained mildly jaundiced for several weeks with a total bilirubin level of 8.2 mg per dl and a direct fraction of 3.6 mg per dl at three weeks following delivery. At six weeks, the total bilirubin value was 2.2 mg per dl with a direct

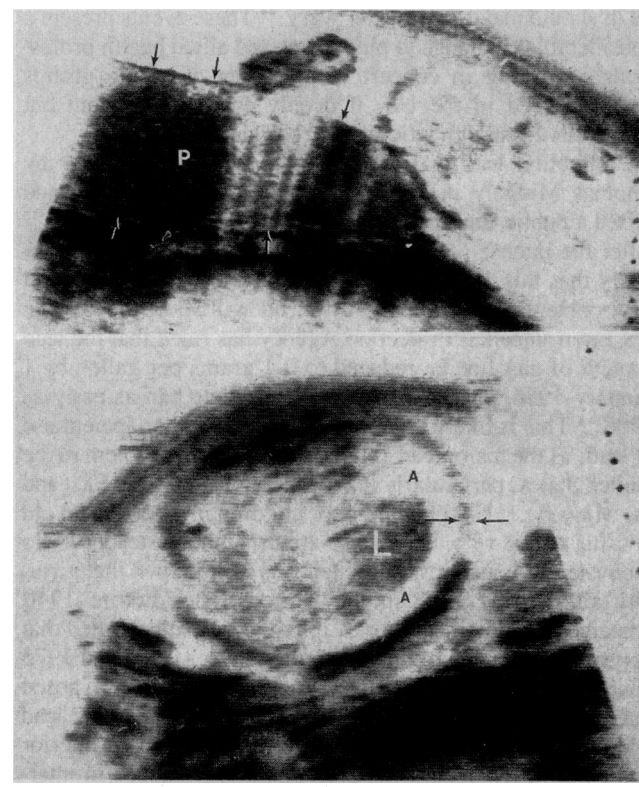

Figure 1.—Top, A longitudinal ultrasound scan shows pronounced placental (P) thickening (8 cm) and polyhydramnios. The margins of the placenta are indicated by arrows. **Bottom,** A transverse scan of the fetal abdomen shows moderate ascites (A). The liver (L) and bowel are outlined by fluid. The thickened abdominal wall is indicated by arrows.

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fraction of 0.7 mg per dl. At about 2½ years of age, the infant is developing normally.

Discussion

With the continued decline in the incidence of rhesus isoimmunization in the obstetrical population, nonimmunologic causes currently account for most cases of hydrops fetalis. Because of the diverse associations among maternal disease states, fetal congenital anomalies and nonimmune hydrops fetalis, uniform diagnostic criteria and a single unifying pathophysiologic concept have not developed.[1] However, most cases variably present with ultrasound evidence of polyhydramnios, placental thickening, edema of the fetal skin and effusions in the abdominal, pleural and pericardial cavities. Overall, perinatal survival is poor, ranging from 20% to 50%. Maternal pregnancy-induced hypertension when associated with nonimmune hydrops fetalis may be severe, frequently occurs before midpregnancy and is associated with a poor fetal outcome.[2] Although unusual, recurrences of hydrops fetalis in the same patient have been reported. Benign transitory ascites has been reported previously.[3]

The case reported here is unusual in that the hydrops fetalis spontaneously regressed in utero despite sonographic features of more advanced nonimmune hydrops fetalis, which in the past has been associated with a poorer fetal prognosis. It is interesting to speculate that the fetus suffered a congenital viral infection with transient myocarditis, hepatitis and congestive heart failure. This in turn resulted in an increase in placental mass with the appearance of the pregnancy-induced hypertension. As the fetal cardiovascular difficulties cleared, the pregnancy-induced hypertension resolved in the mother. Though the fetus was able to clear the cardiovascular component in utero, complete resolution of the hepatitis required an additional six weeks postpartum.

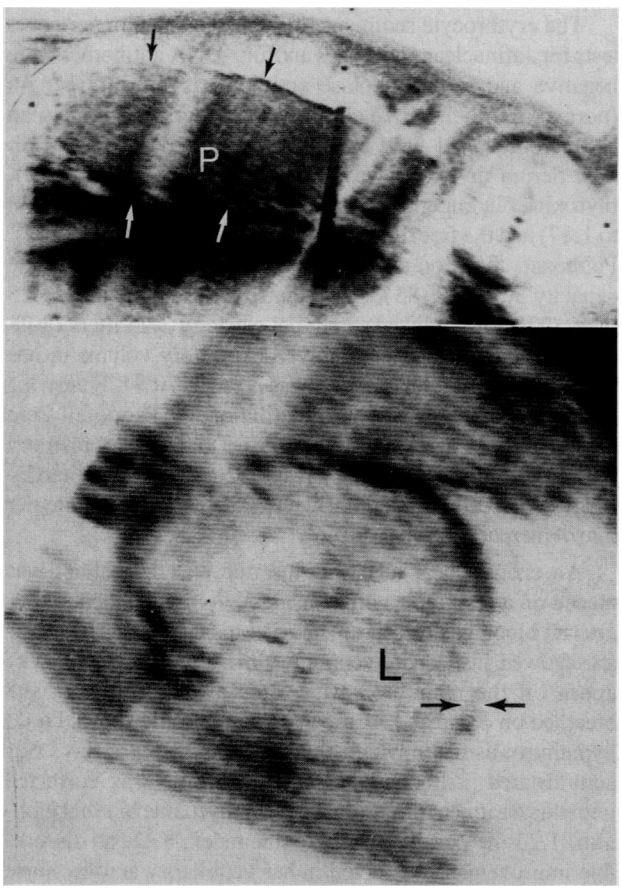

Figure 2.—Top, Scans done two weeks later show a decrease in placental (P) thickness from 8 cm to 6 cm. The margins of placenta are indicated by arrows. **Bottom,** A transverse scan of the fetal abdomen shows resolution of fetal ascites and a decrease in abdominal wall thickness. The abdominal wall is indicated by arrows and the liver by L.

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Hypoventilation in a Case of Nonfamilial Parkinson's Disease

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THE CLINICAL SYNDROME of idiopathic central alveolar hypoventilation is characterized by inadequate automatic ventilation, carbon dioxide retention disproportionate to the degree of mechanical abnormalities of the respiratory system (if any), hypoxemia and cor pulmonale.[1-3] This syndrome has been generally described in association with brain-stem disorders such as bulbar poliomyelitis, medullary infarct and cervical cordotomy.[4,5] Recently, a familial syndrome with parkinsonism and alveolar hypoventilation has been described.[6,7]

We describe the case of a patient with nonfamilial parkinsonism who showed hypoventilation, hypoxemia and cor pulmonale and responded to progesterone administration. This observation may suggest a possible association of hypoventilation with parkinsonism.

Report of a Case

The patient, a 59-year-old woman, was admitted to hospital in November 1979 with respiratory failure. Her major symptom was somnolence, present for at least several weeks before admission; the patient also complained of weakness and mild dyspnea on exertion. She had been diagnosed as having mild Parkinson's disease 15 years previously for

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